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**Dictionary of  
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and  
Molecular Biology**  
**3rd Edition**

**Paul Singleton  
Diana Sainsbury**

**JOHN WILEY & SONS, LTD**

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*heteroplasmon*) contains the nucleus of one parent cell but may contain cytoplasmic elements from both parent cells.

**cytofluorometry** FLOW CYTOMETRY which involves the detection of specific fluorescence or FLUOROCHROME markers.

**cytogamy** (selfing) (*ciliate protozool.*) The occurrence of AUTOGAMY (instead of conjugation) in each of two ciliates which have paired.

**cytohet** (cytoplasmic heterozygote) A *eukaryotic* cell which is HETEROZYGOUS for one or more CYTOPLASMIC GENES.

**cytokines** In the human and animal body: a heterogeneous population of (glyco)proteins which form a dynamic network of intercellular messenger molecules that regulate various aspects of physiology, including the immune response to infection. (This describes, but does not define, cytokines; workers in the field have not yet indicated the essential difference(s) between those molecules which are currently regarded as cytokines and certain other regulatory molecules – thus precluding a formal definition at the present time.) Cytokines may be distinguished from the (protein) hormones in that (i) a given cytokine may be synthesized by different types of cell and/or may act on different types of cell; (ii) cytokines typically act on target cells near the source of cytokine – although if secreted into the circulatory system they may act on distant cells; (iii) cytokines may cause diverse effects (e.g. when acting on different cells under different conditions); (iv) different types of cytokine may give rise to the same physiological effect; and (v) some cytokines can act as mitogens.

Two sources [Book refs 218 and 226] recognize the following as cytokines: COLONY-STIMULATING FACTORS, INTERFERONS, INTERLEUKINS, cytotoxic agents such as tumour necrosis factor (see TNF), growth factors and CHEMOKINES.

Cytokines are synthesized mainly by leukocytes (white blood cells); some cytokines are synthesized by stationary cells (e.g. endothelial cells). Although most cytokines are soluble (secreted) products, some are, or can be, membrane-associated.

In general, transcription of cytokine-encoding genes is inducible by appropriate exogenous or endogenous stimuli. An example of an exogenous stimulus is the binding of lipopolysaccharide to CD14 receptors on macrophages (see CD14); endogenous stimuli can arise e.g. during viral infection.

On release, cytokines bind to specific receptors on target cells.

Cytokine receptors are divided into a number of families which differ e.g. in structure. (Some of the receptors found on macrophages and T lymphocytes can act as receptors for human immunodeficiency virus (HIV). Cytokine receptors have also been reported to act as binding sites for certain other viruses, including human (alpha) herpesvirus 1.) Interestingly, receptor molecules can be shed from cells; such isolated receptors can e.g. bind to (and antagonize) the corresponding cytokine, or they can bind to other cells – which may then be stimulated by the given cytokine.

The binding of a cytokine to its cognate receptor initiates an intracellular signal, the nature of which depends e.g. on the type of cell and cytokine and on the environmental and intracellular conditions under which binding takes place. Cytokine–receptor binding characteristically results (either directly or indirectly) in the activation of kinase(s) at certain stage(s) within the signalling pathway. In one intracellular signalling pathway (triggered by many of the interleukins), the binding of cytokine leads to activation of a tyrosine kinase of the JAK (Janus kinase) family which, when activated, transmits the signal by phosphorylating a molecule of the so-called 'signal transducers and activators of transcription' (STATs). STATs exist in different forms, and

a given type of STAT relays the signal for only some type(s) of cytokine – thus permitting different cytokines to initiate different signals in the same cell. Phosphorylated STATs form homo- or heterodimers that enter the nucleus and promote transcription of particular gene(s). [STATs: TIBS (2000) 25 496–502.]

As mentioned, a given cytokine may cause diverse effects. For example, the binding of tumour necrosis factor (TNF) to its receptor can result in activation of caspases (and APOPTOSIS) – or e.g. transcription of specific genes through activation of a major transcription factor: nuclear factor- $\kappa$ B (NF- $\kappa$ B). In the latter pathway, the binding of TNF to its receptor promotes phosphorylation (and consequent degradation) of a certain protein (I $\kappa$ B $\alpha$ ) which, by binding to NF- $\kappa$ B, inactivates it; that is, phosphorylation (degradation) of I $\kappa$ B $\alpha$  results in the activation of NF- $\kappa$ B. NF- $\kappa$ B is important e.g. in the development of an inflammatory response; it promotes transcription of a range of genes – including those encoding TNF- $\alpha$ , IL-1 $\beta$  and IL-8.

In general, the binding of cytokines to their receptors may initiate cellular responses that range from secretion (of cytokines etc.), differentiation or proliferation to chemotaxis or apoptosis; currently, many of the signalling pathways in eukaryotic cells are incompletely understood. [Signalling mechanisms in prokaryotes and eukaryotes: Book ref. 218, pp 89–162; Book ref. 226, pp 111–140.]

*Note regarding nomenclature of cytokines.* The name of a cytokine does not necessarily correlate with its primary function(s) in vivo. For example, tumour necrosis factor was initially identified as an anti-tumour agent but is now known to be a central mediator in host defence and inflammation. Again, some interleukins, initially thought to mediate only leukocyte–leukocyte interactions, are now known to involve other types of cell; thus, e.g. interleukin-8 is secreted by endothelial cells and is classified as a chemokine.

*Cytokines as factors in health and (infectious) diseases.* Various roles have been attributed to cytokines in normal physiology and development. However, the contribution of individual cytokines in vivo has been difficult to establish experimentally – not least because of the complexity of the system and the existence of biochemical redundancy among cytokines; hence, in some cases, the function(s) of a cytokine have been inferred from the effects attributed to inherited deficiencies in the synthesis or activity of that cytokine (and/or its receptor). In other cases, the role(s) of cytokines have been inferred from studies on knockout mice in which genes of particular cytokine(s) have been rendered non-functional.

In diseases of microbial aetiology, cytokines typically have protective roles – e.g. in processes such as ANTIBODY FORMATION and INFLAMMATION. However, dysregulation of cytokines (enhanced production, imbalance, inhibition) may be a major factor in pathogenesis.

In some cases, the severity of, or susceptibility to, disease has been found to vary in different individuals according to the nature of the TNF- $\alpha$  gene promoter – particular polymorphisms in the promoter region of the gene being associated with enhanced production of the cytokine; this has been reported in cerebral MALARIA [Nature (1994) 371 508–510] and in ENDO-TOXIC SHOCK [JAMA (1999) 282 561–568]. Again, polymorphisms in the gene encoding IL-1 $\beta$  have been associated with an increased risk of gastric cancer from *Helicobacter pylori* infection [Nature (2000) 404 398–402].

In pyelonephritis caused by *Escherichia coli*, the cytokines IL-6 and IL-8 appear to be prominent – levels of IL-6 correlating

## cytokinesis

with the severity of disease [e.g. PNAS (2000) 97 8829–8835 (8833–8834)].

During infection with *Yersinia*, the pathogen's secreted YopP/YopJ (see VIRULON) down-regulates the pro-inflammatory response – e.g. inhibiting the formation of both TNF- $\alpha$  and IL-8: the mechanism involves inhibition of the kinase that phosphorylates (and inactivates) I $\kappa$ B $\alpha$ , thus inhibiting transcription of NF- $\kappa$ B-dependent genes [PNAS (2000) 97 8778–8783 (8781–8782)].

Viruses may inhibit cytokines in various ways; for example, soluble forms of TNF and IL-8 receptors are encoded by Shope fibroma virus and CMV, respectively. (See also INTERLEUKIN-18.)

(See also DISSEMINATED INTRAVASCULAR COAGULATION; DNA VACCINE; HAEMOPHAGOCYTIC SYNDROME; JARISCH-HERXHEIMER REACTION; LEPROSY; MODULIN; NITRIC OXIDE; P FIMBRIAE; SUPER-ANTIGEN; TOXIC SHOCK SYNDROME.)

Many infections elicit an immunological response in which a particular subset of T LYMPHOCYTES – either Th1 or Th2 – is dominant. For example, Th1-type responses are typical in certain bacterial and protozoan infections, while Th2-type responses are common e.g. when infection involves helminths [IT (1996) 17 138–146]; moreover, in at least some cases, an 'inappropriate' response (e.g. Th2 instead of Th1) is associated with increased susceptibility to the infection.

Naive CD4<sup>+</sup> T cells (i.e. those not previously exposed to antigen) may develop as either Th1 or Th2 cells on exposure to antigen; the mechanism that selects one or other subset of T cells is unknown, but the decision to develop one way or the other is influenced by the microenvironment of cytokines which are present during activation of the T cell by antigen – e.g. IL-12 promotes the Th1 response, while IL-4 promotes differentiation to Th2 cells. (A newly reported cytokine receptor, designated TCCR, appears to be necessary for the Th1-type immune response in mice [Nature (2000) 407 916–920].) Interestingly, glucocorticoids from stress metabolism may suppress IL-12 (the main inducer of the Th1-type response); that is, stress-derived glucocorticoids may shift the balance of the immune response from Th1-type toward Th2-type [BCEM (1999) 13 583–595].

One important difference between Th1- and Th2-type responses is that Th1 and Th2 cells secrete different types of cytokine (see table) and so have correspondingly dissimilar physiological roles. (In this context, it is interesting to note that when the molecule ICAM-1 was co-expressed with antigen (on the antigen-presenting cell) the Th2 cytokine IL-4 was found

to be down-regulated in naive CD4<sup>+</sup> cells [PNAS (1999) 96 3023–3028].)

Studies carried out in vitro and in vivo suggest that the cytokines secreted by (Th1 or Th2) cells are responsible for various aspects of the immune response observed during infections. Thus, 'inflammatory' Th1 cells secrete pro-inflammatory cytokines that are associated with important roles in cell-mediated immunity. For example, IFN- $\gamma$  activates macrophages which may then (i) exhibit enhanced antimicrobial activity in phagosomes, (ii) secrete IL-8, attracting immune cells to the site of infection, and (iii) secrete IL-12, promoting further development of the Th1 subset. Moreover, IFN- $\gamma$  can promote class switching to complement-fixing, opsonizing antibodies (human IgG1, IgG3; murine IgG2a). Th1 cytokines can e.g. upregulate expression of E selectins (see INFLAMMATION) and they can also upregulate MHC class II antigens. DELAYED HYPERSENSITIVITY is one manifestation of the characteristically cell-mediated Th1-type immune response.

Th2 cells are typically T-helper cells in ANTIBODY FORMATION. Th2 (anti-inflammatory) cytokines down-regulate macrophages and promote B cell activation, thus being important in the 'humoral' (antibody-mediated) immune response to infection by e.g. extracellular bacteria and helminths etc. IL-4 promotes class switching to non-complement-fixing IgG (human IgG2, IgG4; murine IgG1) as well as to IgE in both man and mice. IL-5 promotes eosinophilia which may give activity against e.g. helminths and other parasites. IL-6 may contribute to antimicrobial activity by stimulating B cell proliferation and/or inducing ACUTE-PHASE PROTEINS.

*Therapeutic uses of cytokines.* Because cytokines regulate so many aspects of the immune defence system they are attractive as candidate therapeutic agents; for example, INTERFERONS have been useful in various contexts. However, some cytokines (e.g. TNF), though potentially useful, may be unsuitable for therapy owing e.g. to instability or toxicity in vivo; interestingly, a non-toxic TNF-mimetic peptide has been found to prevent recrudescence of *Mycobacterium bovis* (BCG) infection in CD4<sup>+</sup> T cell-depleted mice [JLB (2000) 68 538–544]. Inhibition of TNF- $\alpha$  can be achieved by monoclonal antibodies or by soluble TNF- $\alpha$  receptor molecules. [New perspectives on the design of cytokines and growth factors: TIBtech. (2000) 18 455–461.]

[Molecular biology of the cytokines: Book ref. 226.]

**cytokinesis** Those events, excluding nuclear division, which occur during the division of a eukaryotic cell into progeny cells; they include the apportionment of the cytoplasm and organelles, and may include e.g. synthesis of new material for the cell wall of each progeny cell.

**cytokinins** (kinins, phytokinins) PHYTOHORMONES which stimulate metabolism and cell division; the cytokinins are 6-*N*-substituted adenines which are synthesized mainly at the root apex and translocated via the xylem. (Cytokinin-like activity is exhibited by certain urea derivatives, e.g. diphenylurea (found in coconut milk); it is believed that such compounds act by promoting the 6-*N*-substitution of endogenous adenine.) The mechanism by which cytokinins promote cell division is unknown: one suggestion is that cell division is encouraged by an increase in the level of endogenous cAMP brought about by the inhibition of cAMP phosphodiesterase by cytokinins.

Compounds similar or identical to cytokinins are produced by certain microorganisms; such compounds may account for the formation of ROOT NODULES and for the development of symptoms in e.g. FASCINATION.

CYTOKINES: some of the cytokines secreted by Th1 and Th2 subsets of T lymphocytes

Cytokine	Th1	Th2
Interleukin-2	+	
Interleukin-3	+	+
Interleukin-4		+
Interleukin-5		+
Interleukin-6		+
Interleukin-10		+
Interleukin-13		+
Interferon- $\gamma$	+	
TNF- $\alpha$	+	+ <sup>a</sup>
TNF- $\beta$	+	

<sup>a</sup> Secretion from Th2 cells reported to be lower than that from Th1 cells.

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**cy-to-chrome** (sī'tō-krōm). A class of hemoprotein whose principal biologic function is electron and/or hydrogen transport by virtue of a reversible valency change of the heme iron. C's are classified in four groups (*a*, *b*, *c*, and *d*) according to spectrochemical characteristics; many variants exist, particularly among bacteria and in green plants and algae, one being a variant of the *c* type cytochrome called cytochrome *f*. The mitochondrial system of *c*'s provides electron transport through cytochrome *c* oxidase to molecular oxygen as the terminal electron acceptor (respiration). [cyto- + *G. chrōma*, color]

**cy-to-chrome *aa*<sub>3</sub>**. SYN cytochrome *c* oxidase.

**cy-to-chrome *b***. A cytochrome of the respiratory chain. A deficiency of this cytochrome leads to chronic granulomatous disease.

**cy-to-chrome *b*<sub>5</sub>**. A cytochrome in the endoplasmic reticulum that acts with a number of oxygenases; a deficiency of this cytochrome results in a form of hereditary methemoglobinemia.

**cy-to-chrome *b*<sub>5</sub> re-duc-tase**. A flavoenzyme catalyzing the reduction of 2ferricytochrome *b*<sub>5</sub> to 2ferrocycytochrome *b*<sub>5</sub> at the expense of NADH; has a role in fatty acid desaturation; a deficiency can lead to hereditary methemoglobinemia (type I, only observed in erythrocyte cytosol; type II, deficiency in all tissues; type III, deficiency in all hematopoietic cells).

**cy-to-chrome *c***. The mobile cytochrome that transports electrons from Complex III to Complex IV of the respiratory chain.

**cy-to-chrome *cd***. SYN cytochrome oxidase (*Pseudomonas*).

**cy-to-chrome *c*<sub>3</sub> hy-dro-gen-ase**. A hydrogenase enzyme catalyzing reduction of 2ferricytochrome *c*<sub>3</sub> by H<sub>2</sub> to 2ferrocycytochrome *c*<sub>3</sub> and 2H<sup>+</sup>.

**cy-to-chrome *c* ox-i-dase**. A cytochrome of the *a* type, containing copper, that catalyzes the oxidation of 4ferrocycytochrome *c* by molecular oxygen to 4ferricytochrome *c* and 2H<sub>2</sub>O. A part of Complex IV of the respiratory chain. A deficiency of one or more of the polypeptides of this complex results in neuronal loss in brain leading to psychomotor retardation and neurodegenerative disease. SYN cytochrome *aa*<sub>3</sub>, indophenol oxidase, indophenolase.

**cy-tō-chrome *c* re-duc-tase**. SYN NADH dehydrogenase.

**cy-to-chrome *c*<sub>2</sub> re-duc-tase**. SYN NADPH-cytochrome *c*<sub>2</sub> reductase.

**cy-to-chrome ox-i-dase (*Pseu-do-mo-nas*)**. An enzyme with action identical to that of cytochrome *c* oxidase, but acting on ferrocycytochrome *c*<sub>2</sub>. SYN cytochrome *cd*.

**cy-to-chrome P-450<sub>SCC</sub>**. Cholesterol monooxygenase (side chain cleaving). [450 nm, the absorption maximum that the reduced cytochrome complexed with carbon monoxide exhibits]

**cy-to-chrome per-ox-i-dase**. A hemoprotein enzyme catalyzing the reaction between H<sub>2</sub>O<sub>2</sub> and 2ferrocycytochrome *c* to yield 2ferricytochrome *c* and 2H<sub>2</sub>O.

**cy-to-chrome re-duc-tase**. SYN NADPH-ferrihemoprotein reductase.

**cy-to-chy-le-ma** (sī'tō-kī-lē'mā). The more fluid portion of the cytoplasm. [cyto- + *G. chylos*, juice]

**cy-toc-i-dal** (sī'tō-sī'dāl). Causing the death of cells. [cyto- + *L. caedo*, to kill]

**cy-to-cide** (sī'tō-sīd). An agent that is destructive to cells. [cyto- + *L. caedo*, to kill]

**cy-toc-la-sis** (sī-tok'lā-sis). Fragmentation of cells. [cyto- + *G. klasis*, a breaking]

**cy-to-clas-tic** (sī-tō-klas'tik). Relating to cytoclasis.

**cy-to-cle-sis** (sī-tō-klē'sis). The influence of one cell on another. SYN biotaxis (2), cytotaxis. [cyto- + *G. klēsis*, a call]

**cy-to-cu-pre-in** (sī-tō-koo'prē-in). Former terms for copper-containing proteins found in human erythrocytes and other tissues. SEE superoxide dismutase, ceruloplasmin. SYN cerebropuprein, erythropuprein, hemocuprein, hepatocuprein.

**cy-to-cyst** (sī'tō-sist). Rarely used term for the bladderlike remains of the red blood cell or tissue cell that encloses a mature schizont. [cyto- + *G. kystis*, bladder]

**cy-to-di-ag-no-sis** (sī'tō-dī-ag-nō'sis). Diagnosis of the type and, when feasible, the cause of a pathologic process by means of

microscopic study of cells in an exudate or other form of body fluid.

**cy-to-di-er-e-sis** (sī'tō-dī-er'ē-sis). SYN cytokinesis. [cyto- + *G. diairesis*, division]

**cy-to-gene** (sī'tō-jēn). SYN plasmagene.

**cy-to-gen-e-sis** (sī-tō-jen'ē-sis). The origin and development of cells. [cyto- + *G. genesis*, origin]

**cy-to-ge-net-i-cist** (sī'tō-jē-net'i-sist). A specialist in cytogenetics.

**cy-to-ge-net-ics** (sī'tō-jē-net'iks). The branch of genetics concerned with the structure and function of the cell, especially the chromosomes.

Cytogenetics arose as a fusion of 19th century cytology and 20th century genetics, which came into being in 1903 with the articulation of the chromosome theory of inheritance. The developing field concerned itself with detailing the behavior of chromosomes and their functional subunits, the genes, during reproduction, and with relating that behavior statistically to characteristics of the resulting cells or animals. Modern molecular cytogenetics involves the microscopic study of chromosomes that have been fixed in mitosis and stained with various agents to delineate characteristic bands. DNA probes can be applied to locate specific gene sequences. Karyotyping is the arrangement of photographs of stained chromosomes in a standard format. Cytogenetic techniques are used to test for inborn errors of metabolism and genomic aberrations such as Down syndrome and to determine sex in cases where anatomy is inconclusive.

**cy-to-gen-ic** (sī-tō-jen'ik). Relating to cytogenesis.

**cy-tog-e-nous** (sī-toj'ē-nūs). Cell-forming.

**cy-to-glu-co-pe-nia** (sī'tō-gloo-kō-pē'nē-ā). An intracellular deficiency of glucose. [cyto- + glucose + *G. penia*, poverty]

**cy-toid** (sī'toyd). Resembling a cell. [cyto- + *G. eidos*, resemblance]

**cy-to-ker-a-tin** (sī-to-ker-a-tinz). SYN keratin.

**Cy-to-kine** (sī'tō-kīn). Any of numerous hormonelike, low-molecular-weight proteins, secreted by various cell types, that regulate the intensity and duration of immune response and mediate cell-cell communication. SEE interferon, interleukin, lymphokine, chemokines. See entries under various growth factors. SEE ALSO interferon, interleukin, lymphokine. [cyto- + *G. kinēsis*, movement]

Cytokines are produced by macrophages, B and T lymphocytes, mast cells, endothelial cells, fibroblasts, and stromal cells of the spleen, thymus, and bone marrow. They are involved in mediating immunity and allergy and in regulating the maturation, growth, and responsiveness of particular cell populations, sometimes including the cells that produce them (autocrine activity). A given cytokine may be produced by more than one type of cell. Some cytokines enhance or inhibit the action of other cytokines. The first cytokines to be identified were named according to their functions (e.g., T cell growth factor), but this nomenclature became awkward because several cytokines can have the same function, and the function of a cytokine can vary with the circumstances of its elaboration. Later, as the chemical structure of each cytokine was determined, it was designated an interleukin and assigned a number (e.g., interleukin-2 [IL-2], formerly T cell growth factor). Cytokines have been implicated in the generation and recall of long-term memory and the focusing of attention. Some of the degenerative effects of aging may be due to a progressive loss of regulatory capacity by cytokines. Because cytokines derived from the immune system (immunokines) are cytotoxic, they have been used against certain types of cancer.

**c. network**, a key cellular function.

**cy-to-ki-ne-sis**. plasm of the cytodieresis. [cy-

**cy-to-lem-ma** (sī-tō-lem-ma, husk)

**cy-to-lip-in** (sī-tō-lip-in). mide oligosacch

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IL-1α 17

IL-1β 17

IL-2 15

IL-3 14

IL-4 15

IL-5 45

IL-6 26

IL-7 20

IL-9 32

IL-10 35

IL-11 23

IL-12 35, 4

IL-13 9-1

IL-14 60

IL-15 14

IL-16 17

IL-17 30-3

IL-18 18

G-CSF 18-2

M-CSF 45-9

GM-CSF 22

TGF-β 25

IFN-γ 40-7

TNF-α 17

LT-α (TNF-β) 20-2

IF 46

Generally, an aqueous s. of a nonvolatile substance. 3. In the language of the Pharmacopeia, an aqueous s. of a nonvolatile substance is called a s. or liquor; an aqueous s. of a volatile substance is a water (aqua); an alcoholic s. of a nonvolatile substance is a tincture (tinctura); an alcoholic s. of a volatile substance is a spirit (spiritus); a s. in vinegar is a vinegar (acetum); a s. in glycerin is a glycerol (glyceritum); a s. in wine is a wine (vinum); a s. of sugar in water is a syrup (syrupus); a s. of a mucilaginous substance is a mucilage (mucilago); a s. of an alkaloid or metallic oxide in oleic acid is an oleate (oleatum). 4. The termination of a disease by crisis. 5. A break, cut, or laceration of the solid tissues. SEE s. of contiguity, s. of continuity. SYN solutio. [L. solutio]

**acetic s.**, a vinegar.

**amaranth s.**, a 1% s. of amaranth (trisodium naphthol sulfonic acid), a synthetic vivid red dye, stable in acid and intensified in sodium hydroxide s.; used as a red or pink colorant in liquid pharmaceuticals.

**aqueous s.**, a s. containing water as the solvent; examples include lime water, rose water, saline s., and a large number of s.'s intended for intravenous administration.

**Benedict s.**, an aqueous solution of sodium citrate, sodium carbonate, and copper sulfate which changes from its normal blue color to orange, red, or yellow in the presence of a reducing sugar such as glucose. SEE ALSO Benedict test for glucose.

**Burow s.**, a preparation of aluminium subacetate and glacial acetic acid, used for its antiseptic and astringent action on the skin.

**chemical s.**, SEE solution (1).

**colloidal s.**, a dispersoid, emulsoid, or suspensoid. SYN colloidal dispersion.

**s. of contiguity**, the breaking of contiguity; a dislocation or displacement of two normally contiguous parts.

**s. of continuity**, division of bones or soft parts that are normally continuous, as by a fracture, a laceration, or an incision. SYN dieresis.

**Dakin s.**, a bactericidal wound irrigant. SYN Dakin fluid.

**disclosing s.**, a s. that selectively stains all soft debris, pellicle, and bacterial plaque on teeth; used as an aid in identifying bacterial plaque after rinsing with water.

**Earle s.**, a tissue culture medium containing  $\text{CaCl}_2$ ,  $\text{MgSO}_4$ , KCl,  $\text{NaHCO}_3$ , NaCl,  $\text{NaH}_2\text{PO}_4 \cdot \text{H}_2\text{O}$ , and glucose.

**ethereal s.**, a s. of any substance in ether.

**Fehling s.**, an alkaline copper tartrate s. formerly used for detection of reducing sugars. SYN Fehling reagent.

**ferric ammonium acetate s.**, a clear, aromatic, reddish-brown liquid which has been used in iron-deficiency anemia in animals and man; a source of iron. SYN Basham mixture.

**Fonio s.**, a diluent with magnesium sulfate, used for stained smears of blood platelets.

**Gallego differentiating s.**, a dilute s. of formaldehyde and acetic acid used in a modified Gram stain to differentiate and enhance the basic fuchsin binding to Gram-negative microorganisms.

**Gey s.**, a salt s. usually used in combination with naturally occurring body substances (e.g., blood serum, tissue extracts) and/or more complex chemically defined nutritive s.'s for culturing animal cells.

**Hanks s.**, a salt s. usually used in combination with naturally occurring body substances (e.g., blood serum, tissue extracts) and/or more complex chemically defined nutritive s.'s for culturing animal cells; two variations contain  $\text{CaCl}_2$ ,  $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$ , KCl,  $\text{KH}_2\text{PO}_4$ ,  $\text{NaHCO}_3$ , NaCl,  $\text{Na}_2\text{HPO}_4 \cdot 2\text{H}_2\text{O}$ , and D-glucose.

**Hartman s.**, a s. used to desensitize dentin in dental operations; contains thymol, ethyl alcohol, and sulfuric ether.

**Hartmann s.**, SYN lactated Ringer s.

**Hayem s.**, a blood diluent used prior to counting red blood cells.

**Krebs-Ringer s.**, a modification of Ringer s., prepared by mixing NaCl, KCl,  $\text{CaCl}_2$ ,  $\text{MgSO}_4$ , and phosphate buffer, pH 7.4.

**lactated Ringer s.**, a s. containing NaCl, sodium lactate,  $\text{CaCl}_2$  (dihydrate), and KCl in distilled water; used for the same purposes as Ringer s. SYN Hartmann s.

**Lange s.**, a colloidal gold s. used to demonstrate protein abnormalities in spinal fluid. SEE Lange test.

**Locke s.'s**, s.'s containing, in varying amounts, NaCl,  $\text{CaCl}_2$ , KCl,  $\text{NaHCO}_3$ , and D-glucose; used for irrigating mammalian heart and other tissues, in laboratory experiments; also used in combination with naturally occurring body substances (e.g., blood serum, tissue extracts) and/or more complex chemically defined nutritive s.'s for culturing animal cells.

**Locke-Ringer s.**, a s. containing NaCl,  $\text{CaCl}_2$ , KCl,  $\text{MgCl}_2$ ,  $\text{NaHCO}_3$ , D-glucose, and water; used in the laboratory for physiological and pharmacological experiments.

**Lugol iodine s.**, an iodine-potassium iodide s. used as an oxidizing agent, for removal of mercurial fixation artifacts, and also in histochemistry and to stain amebae.

**molecular dispersed s.**, SYN dispersoid.

**Monseil s.**, ferric subsulfate s. used to coagulate superficial bleeding such as that following skin biopsy.

**normal s.**, SEE normal (3).

**ophthalmic s.'s**, sterile s.'s, free from foreign particles and suitably compounded and dispensed for instillation into the eye.

**Ringer s.**, (1) a s. resembling the blood serum in its salt constituents: it contains 8.6 g of NaCl, 0.3 g of KCl, and 0.33 g of  $\text{CaCl}_2$  in each 1000 mL of distilled water; used as a fluid and electrolyte replenisher by intravenous infusion. (2) a salt s. usually used in combination with naturally occurring body substances (e.g., blood serum, tissue extracts) and/or more complex chemically defined nutritive s.'s for culturing animal cells. SYN Ringer lactate. SEE Ringer injection.

**saline s.**, (1) a s. of any salt; SYN salt s. (2) specifically, an isotonic sodium chloride s.; 0.85–0.9 per 100 mL of water.

**salt s.**, SYN saline s. (1).

**saturated s. (sat. sol., sat. soln.)**, a s. that contains all of a substance capable of dissolving; a solution of a substance in equilibrium with an excess undissolved substance.

**standard s., standardized s.**, a s. of known concentration, used as a standard of comparison or analysis.

**supersaturated s.**, a s. containing more of the solid than the liquid would ordinarily dissolve; it is made by heating the solvent when the substance is added, and on cooling the latter is retained without precipitation; addition of a crystal or solid of any kind usually results in precipitation of the excess solute, leaving a saturated s.

**test s.**, a s. of some reagent, in definite strength, used in chemical analysis or testing.

**Tyrod s.**, a modified Locke s.; it contains 8 g of NaCl, 0.2 g of KCl, 0.2 g of  $\text{CaCl}_2$ , 0.1 g of  $\text{MgCl}_2$ , 0.05 g of  $\text{NaH}_2\text{PO}_4$ , 1 g of  $\text{NaHCO}_3$ , 1 g of D-glucose, and water to make 1000 mL; used to irrigate the peritoneal cavity, and in laboratory work.

**volumetric s. (VS)**, a s. made by mixing measured volumes of the components.

**Weigert iodine s.**, an iodine-potassium iodide mixture used as a reagent to alter crystal and methyl violet so that they are retained by certain bacteria and fungi.

**sol-vate (sol'vāt)**. A nonaqueous solution or dispersoid in which there is a noncovalent or easily reversible combination between solvent and solute, or dispersion means and disperse phase: when solvent is the solvent or dispersion medium, it is called a hydrate.

**sol-va-tion (sol-vā'shūn)**. Noncovalent or easily reversible combination of a solvent with solute, or of a dispersion means with the disperse phase; if the solvent is water, s. is called hydration. S. affects the size of ions in solution, thus  $\text{Na}^+$  is much larger in  $\text{H}_2\text{O}$  than in solid NaCl.

**sol-vent**. A liquid that holds another substance in solution, i.e., dissolves it. [L. solvens, pres. p. of solvo, to dissolve]

**amphiprotic s.**, a s. capable of acting as an acid or a base: e.g.,  $\text{H}_2\text{O}$ . SEE solvolysis.

**fat s.'s**, organic liquids notable for their ability to dissolve lipids; usually, but not always, immiscible in water; e.g., diethyl ether, carbon tetrachloride. SYN nonpolar s.'s.

**nonpolar s.'s**, SYN fat s.'s.

**polar s.'s**, s.'s that exhibit polar forces on solutes, due to high

dipole moment  
e.g., water, alcohol

**universal s.**, a s. by some to have substances; so

**sol-vol-y-sis (sō'vol-y-sis)**, the solvent to neutralization.

called hydrolysis.

**so-ma (sō'mā)**, trunk, and tail, exception of nerve cell, from body]

**so-man (sō'mān)**, SEE ALSO sari

**so-mas-the-n**

**somat-**, SEE

**so-ma-tag-nō** [somat- + G.

**so-ma-tal-gia**, organic cause

algos, pain]

**so-ma-tas-the**, physical weak

G. asthenia, v

**so-ma-tes-the**, conscious aware

aisthēsis, sensa

**so-mat-es-the**, the body cavity

Relating to or

cle and the inn

visceral (invol

parietal (3). 3.

the generative.

**so-mat-i-co-spl**, the body and t

relating to the

**so-mat-i-co-vis**, splanchnic.

**so-ma-tist (sō'mā-tist)**, neuroses and p

**so-ma-ti-za-tion**, biological need

expression or c

wish for materi

injury, or a re

disorder.

**somato-**, soma

body]

**so-ma-to-chron**, rons or nerve c

completely sur

**so-ma-t-o-crin-i**, leasing hormon

**so-ma-to-g-nic**, body under the

body cells. [son

**so-ma-to-lib-**, r

the hypothalam

hormone (soma

growth hormon

tor, somatotropi

to free, + -in]

**so-ma-to-log-y**, study of the b

[somato- + G. l

**so-ma-to-mam-**, hormone, closel